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A review of neck injury and protection in vehicle accidents

Fan Li¹, Niansong Liu¹, Honggeng Li^{1,*}, Biao Zhang¹, Shiwei Tian¹, Minggang Tan¹ and Baptiste Sandoz²

¹State Key Laboratory of Advanced Design and Manufacturing for Vehicle Body, Hunan University, No. 2, Lushan South Road, Yuelu District, Changsha 410082, China

²Arts et Metiers ParisTech, IBHGC, 151 bd de l'Hopital 75013 Paris, France

*Corresponding author. E-mail: honggengli@hnu.edu.cn

Abstract

Neck injury is one of the most common types of injury in vehicle accidents. The mechanisms of neck injury remain controversial due to the complex structure of the cervical spine and various impact conditions. The aim of the present study is to provide a summary of recent research on neck injury mechanisms, neck injury criteria and neck injury prevention measures. The main types of neck injury resulting from vehicle accidents, including whiplash injury, cervical bone fractures and spinal cord injury, are introduced. Neck injury mechanisms are summarized according to load directions, test or simulation methods, and thresholds by means of impact intensity, load intensity and stress/strain conditions. Neck injury criteria are introduced, including NIC, N_{ij} , N_{km} and LNL. Passive and active technologies for neck injury prevention are described and the challenge of neck injury prevention for future intelligent vehicles is discussed.

Keywords: neck injury; whiplash; cervical fracture; spinal cord injury; neck protection; vehicle accidents

1. Introduction

Traffic accidents are the main cause of neck injuries that often result in long-term suffering and great socio-economic cost. More than 800 000 cases of vehicle crashes involving neck injuries are reported in the United States annually, and the cost of treatment is as high as \$5.2 billion [131].

Neck injuries vary in severity and type. Neck injuries run the gamut from mild to life-altering

or even lethal, as the impact load or inertia of the neck increases in traffic accidents. Whiplash may result in one or several diagnoses such as ligament sprain, disc injury, and muscle strain. A herniated disc or vertebral dislocation in the cervical spine may irritate spinal nerve roots or, more rarely, the spinal cord, causing nerve symptoms.

This paper summarizes the available literature on neck injuries resulting from vehicle accidents—including whiplash injuries, neck fractures

and cervical spinal cord injuries—according to injury type, mechanism, criteria and prevention. Whiplash injuries include skin, muscle, blood vessels, nerves, ligaments and intervertebral discs injuries; neck fractures, meanwhile, comprise vertebral body and joint damage. Spinal cord injuries are discussed separately, as their injury mechanisms differ considerably from those of other forms of soft-tissue damage.

1.1 Whiplash injuries (soft-tissue injuries)

Whiplash often occurs in low-velocity rear-end vehicle collisions. In the United States, 90% of injuries in rear-end crashes are neck injuries [103]. Traffic accident data compiled in Germany reveals that over 90% of whiplash injuries result from rear impacts at speeds of less than 25 km/h [134]. Rear-end collisions are associated with the highest relative risk of whiplash injury when compared with lateral impacts, while drivers show the strongest association with respect to seating position when compared with passengers in the rear seats [10]. More women than men suffer from whiplash (1.5:1) [100], and also suffer long-term symptoms more frequently (odds ratio: 1.54) [19]. The annual incidence among adolescents and adults is 3–6% [122].

In rear-end impact accidents, cervical vertebrae usually suffer a sudden forceful hyperextension followed by hyperflexion. Cervical soft tissues suffer sprains or strains in this period, leading to a variety of clinical symptoms, such as headaches, dizziness, forgetfulness and emotional/psychological disturbances [121]. While some individuals recover from initial symptoms within a few weeks after the accident, up to 50% develop persistent symptoms [18]. These symptoms are called whiplash-associated disorders (WAD). There are several possible sites for this injury: facet joints, spinal ligaments, intervertebral discs, vertebral arteries, dorsal root ganglia and neck muscles [23, 116].

The Quebec Task Force team has classified whiplash injuries according to the severity of the clinical symptoms, from WAD 1 to WAD 5, as shown in Table 1 [19]. The anterior longitudinal ligament is often pulled and injured, which is defined as WAD 1 [57]. A combination of lateral bending and torsion results in unilateral facet dislocations or unilateral locked facets, classified as WAD 2 [83]. WAD 3 comprises common disc injuries, such as ‘edge injuries’ or transverse tearing near the anterior edge of the intervertebral

Table 1. AD classification levels

Injury level	Clinical manifestation
1	No neck discomfort, no abnormal signs
2	Neck pain, stiffness or only tenderness, no abnormal signs
3	Neck symptoms and musculoskeletal signs
4	Neck symptoms and neurological signs
5	Neck symptoms and fractures or dislocations

disc [28]. During the posterior collision, the anterior and posterior cervical muscles are stretched and injured, with the resulting injury ranging from WAD1 to WAD3 according to its severity [16]. However, neck muscle active force affects the dynamic response of the head and neck and the risk of whiplash during a collision, which is more pronounced in low-speed collisions [71]. When extra force is applied to the nerve by surrounding tissues, such as nerve impingement, bone, cartilage, muscles and tendons, nerve function may be affected, leading to pain, numbness, weakness and tingling (WAD 2) [1].

1.2 Neck fractures (bone injuries)

Cervical fracture is characterized by vertebral fracture, and most serious neck fractures are generally accompanied by a cervical dislocation, which may cause spinal instability. In cases of severe dislocation, the cervical bone may fully displace forward (a complication referred to as ‘jumping’) and lock in this position, which makes the ligaments rupture completely. Dislocations may damage the spinal cord and normally require surgery. Neck fractures are usually AIS 2+ injuries.

The angle of force acting during impact often affects the type and severity of the cervical fracture. The injury mode of the cervical vertebra depends on the load direction, which can be divided into compression of the neck, tension, shear, flexion moment, extension moment and axial torsion [79]. Injuries caused by tensile load include dislocation of the occipital condyles, ligamentous injury and fractures [128]. The combination of the bending moment of the neck and the axial compression force can cause a wedge fracture of the anterior vertebral body. An increase of force may cause a burst fracture or bilateral facet dislocation [79]. Table 2 summarizes the characteristics of various cervical spine injuries with their AIS grades.

A pedestrian or vehicle occupant who blocks with his/her head is at high risk of cervical fracture.

Table 2. Characteristics of cervical spine injuries, with AIS grades

Injury category	AIS	Load form	Reference
Jefferson fracture, hangman's fracture	2+	Compression	(Viano D. [130])
Dislocation of the occipital condyles, ligamentous injury and fractures	1+	Tensile load	[128]
Wedge fracture of the anterior vertebral body	2+	Compression-flexion	[79]
Fractures	2+	Compression-extension	(Frank A. Pintar [97])
Bilateral facet dislocation	2	Tension-flexion	[79]
Hangman's fracture, damage to soft tissue	3	Tension-extension	(Viano D. [130])
Facet dislocations or unilateral locked facets	2	Lateral bending and compression	[83]

Elderly people with osteoporosis are at higher risk because of the fragility of their bones. Müller et al. reported that in Germany, cervical spine injuries accounted for 45% of all vertebral fractures in traffic accidents, of which 43% of neck injuries occur in multiple collisions [85]. Sidon et al. stated that men were more likely to suffer cervical spine injuries in car crashes than women (80:29) [113]. Cervical spine injuries in children accounted for a higher proportion of vertebral injuries, reaching 75% [98], and also resulted in a higher mortality rate [35].

1.3 Cervical spinal cord injuries (CSCI)

Spinal cord injury (SCI), a highly disabling and often fatal injury (usually an AIS 3+ injury), occurs when a fracture, dislocation or other neck injury damages the spinal cord [138]. SCI involves sudden forceful damage to the spinal nerves, resulting in temporary or permanent paralysis, bladder and bowel dysfunction and autonomic imbalance, among other consequences [11, 47]. A person with SCI is at immediate risk of respiratory and cardiac failure, which may lead to death in the acute phase [118]. According to numerous investigations, traffic accidents have become the second leading cause of spinal cord injuries [37].

The causes and damage of CSCI are diverse. Atlantoaxial instability results in upper spinal cord injury [109]. Traffic accidents can cause disc herniation [30], ligament injury [30, 146] and fracture and dislocation of the spine [93], which can cause spinal canal narrowing [56, 109], followed by compression of the cervical spinal cord, resulting in SCI. The rapid extension of the cervical vertebral body in traffic accidents can also cause SCI [110]. If the spinal cord is damaged at the third cervical vertebra or above, the victim may die or need a respirator to stay alive. People living with SCI often endure a lifelong disability with complete or incomplete paralysis below the level of injury. Those who survive the acute phase face a life-

long risk of secondary complications such as pressure ulcers, urinary tract infections, deep venous thrombosis, contractures, chronic pain and spasms [11, 144].

The incidence rate of CSCI tends to decline progressively; the number of male patients is significantly higher than the number of female patients; the mean patient age has increased over time; and the upper cervical levels are currently the most commonly affected. A statistical report from a regional trauma centre in Canada showed that vehicle crashes account for 71% of incidents of CSCI; 66% of CSCI patients are male; 75% are under 50 years of age; and the most common spinal level injured is C2 (27%) followed by C5 (22%) [127]. The levels of spinal injury in individuals vary between occupants of front and rear seats in vehicle crashes. An epidemiology study of traumatic spinal cord injury (TSCI) in Spain over a 20-year period (1995–2014) reported a total of 1195 patients with TSCI, of which 76.4% were male and 23.6% female [84]. The mean patient age was 50.2 years. Traffic accidents accounted for 37% of all TSCI injuries. The mean patient age increased significantly over time (from 46.4 to 56.54 years). The most commonly affected neurological level was the cervical level (54.9%), increasing in the case of levels C1–C4 over time.

2. Neck injury mechanisms and criteria

To explore the mechanisms of neck injuries and to quantify the thresholds for injuries to various tissues, biomechanical tests and simulations using mathematical models are widely used. Biomechanical tests provide valuable data, but some of the internal biomechanical responses of soft tissue can neither be easily measured by experimental techniques nor be measured *in vivo*, and the tests are of bad repeatability considering individual differences. Researchers have used the information gathered from animal

experiments and cadaveric studies to develop and evaluate neck mechanical and digital neck models. Digital models provide a cost-effective alternative experimental method for estimating the internal biomechanical response of soft tissue.

2.1 Whiplash mechanisms

Whiplash usually causes injuries to the soft tissue of the neck. Ligaments and discs are the representative soft tissues in neck injuries. The ligament has the physiological function of stabilizing the intervertebral disc and the vertebral body. For all impact configurations, the spinal levels at greatest risk of ligament and/or disc injury are C3/C4 to C7/T1 [57, 116]. The cervical muscles bear the weight of the head 3/4. During the impact resulting from a traffic accident, especially a low-speed collision, the neck muscles have the effect of stabilizing the position of the head and neck [71]. In addition, the blood vessels and nerves in the neck are dense and can cause serious injury in a high-impact accident.

2.1.1 Muscle injury mechanisms. Symptoms of cervical muscle injury include pain, muscle cramps and decreased flexibility caused by excessive strain. Neck-muscle damage always occurs in eccentric contraction when extension is applied during active contraction. Computer simulations of experimental kinematics using human subjects exposed to rear-end collisions have shown that the anterior and posterior neck muscles undergo active prolongation during posterior impact [16, 129]. The anterior sternocleidomastoid muscle is active and prolonged during the contraction phase, while the posterior muscle is active and prolonged during the rebound phase [41]. The muscle strain mechanism suggests that muscles could also be used as candidates for soft-tissue damage from posterior impact. However, this hypothesis is inconsistent with the fact that most patients experience pain in the back of the neck, while the anterior muscles are the first to stretch in the event of rear-end impact [20].

The threshold for muscle damage is difficult to judge. Due to ethical limitations and loss of activity in cadaveric sample muscles, studies of the detailed mechanisms of neck muscle damage need to be done by in-vivo animal experiments, or on in vivo human volunteers at low accelerations and then perform extrapolations. Histological evaluation of porcine posterior cervical muscles after a forceful translational and extensional head

retraction simulating high-speed rear end impact has been conducted [40]. Injuries of the deepest posterior neck muscles could be found in this study, especially in the musculus obliquus samples. Further studies need to be conducted to determine whether lower exposure forces also cause these muscle injuries.

2.1.2 Ligament injury mechanisms. Ligaments are strong bands of connective tissue that hold bones together, and ligament injury is usually a sprain. The main ligaments below the axis include the anterior and posterior longitudinal, capsular, interspinous and supraspinous ligaments, and the *ligamentum flavum*. Spinal ligaments can partially or completely rupture when stretched beyond their physiological limit [116]. Sudden twists occurring in vehicle accidents that overload or overstretch one or more joints in the cervical spine cause ligament injury. Symptoms of neck sprain can be very varied, but may include pain at the back of the patient's neck that worsens when the patient moves, pain that comes on slowly and peaks after about 24 hours, a headache at the back of the neck, muscle spasms and pain in the patient's upper shoulder, neck stiffness, and numbness, weakness or tingling in the patient's arm.

Different ligament injuries have different mechanisms due to their different physiological locations and physiological roles. Hyperextension places the anterior longitudinal ligament (ALL) and facet capsule (FC) ligaments at risk, and hyperflexion motion often damages the flaval (LF) and interspinous (ISL) ligaments [57, 95]. Whiplash simulations using cadavers and monkeys have produced ALL tears and anterior disc detachments [74, 139]. Magnetic resonance-imaging studies have demonstrated injuries to both components in whiplash patients, and similar injuries have also been discovered at surgery and autopsy [17, 60]. Combined shear, bending, and compression load levels that occur in rear-end impacts may cause the cervical facet capsular ligaments injuries. [25, 77, 95, 114].

Different ligaments also have different damage thresholds (Table 3). Ivancic et al. found that the greatest ALL strains occurred in the lower cervical spine and that ALL strains increased with impact severity. Increases over the physiological strains were observed in the middle and lower cervical spine (C3–C7) at trauma energies of 3.5 g and above, suggesting that the ALLs spanning these levels were at the greatest risk of injury [57]. Trends are present for the failure force and stiffness of the

Table 3. Summary of cervical ligament injury mechanisms

Mechanism	Site	Methodology	Threshold value	Reference
Elongation	Capsular ligament	Simulation (FE model)	> 3.82 mm	[133]
Strain	ALL	Cadaveric tests	42.6–47.6%	[140]
	ALL	Simulation (in vitro cervical spine)	0.222	[57]
Extension	Alar ligament	Uniaxial mechanical testing in seven specimens	200 N	[34]
	Transverse ligament	Ligamentous injury	350 N	
	Cadaver tests	Functional unit	56.7 N·m	[44]
Shear (a-p)	Ligament rupture	Simulation (FE model)	824 N	[39]
Impact severity	Apical ligament		15.2 g frontal or 11.7 g rear-end impact	[38]
	Alar ligament	Simulation (FE model)	20.7 g frontal or 14.4 g rear-end impact	
	Capsular ligament	Simulation (FE model)	11.3 g frontal or 12.8 g rear-end impact	
	ALL of C3–C7	Simulation (vitro cervical spine)	3.5 g	[57]
Vertebral dislocation	TAL		Lateral displacement > 7 mm	(J. F. [24])
			DAI > 3 mm	[48]

ALL to decrease with increasing age [78]. Using an FE model, Fice, Cronin and Panzer found that in frontal and rear-end impacts, the highest predicted distractions in the upper cervical spine were for the apical and alar ligaments [38]. Linearly interpolating between the predicted distractions for the apical and alar ligaments at different frontal impact accelerations, the model predicted that at 15.2 g and 20.7 g, respectively, the distraction of those ligaments exceeds the average failure distraction minus one standard deviation. Ivancic and Xiao observed nonphysiologic flexion at C7/T1 during a 13.3 g crash, indicating potential tensile injury of the supraspinous, interspinous and capsular ligaments and the *ligamentum flavum*, and compression injury of the anterior disc [58]. They also observed nonphysiologic extension at C6/7 and C7/T1 during head/HR contact, indicating potential tensile injury of the anterior longitudinal ligament and anterior annular fibres, and compression injury of the facet joints.

The transverse ligament (TL) is subject to a special mechanism of injury. Severe injuries to the transverse ligament and the posterior atlanto-occipital membrane were more common in frontal than in rear-end collisions [28]. Cusick and Yoganandan found that a lateral displacement of greater than 7 mm indicates risk of tearing of the insertions of the transverse atlantal ligament (TAL), with resultant risk of atlantoaxial instability [24]. An atlas-dens interval (ADI)—defined as the distance between the anterior portion of the atlas and the dens of the axis—exceeding 3 mm for an adult was found to be an implication of failure of either (or both) the transverse ligament and the alar

ligament [33, 39, 51, 68, 70, 106]. For others, the ADI should not exceed 3 to 3.5 mm [48]. In fact, the ADI is often used to assess trauma to the cervical spine based on its value increase, which can be an indication of TL rupture. The flexion of the head and neck complex is increased by the TL rupture as well as the ADI interval under the compressive load [82].

Muscles play a very important role in protecting ligaments from overloading. The importance of muscle activation has been demonstrated for a 7 g rear-end impact, with Capsular ligament (CL) strain reduced from 28% to 13% with active muscles [38]. Muscle activation leads to peak CL strain reductions of 4%, 15% and 12% for rear-end impacts of 4 g, 7 g and 10 g, respectively.

2.1.3 Disc injury mechanisms. Mechanical overloading from hyperflexion and torsion is considered to be a potential cause of disc failure [5, 6, 36, 143]. A common disc injury is a ‘rim lesion’ or transverse tear near the anterior vertebral rim. This is caused by distraction and shearing in sudden extension. Disc contusion or herniation is often accompanied by facet-joint hematoma, peripheral spinal nerve and spinal cord contusion or articular process fracture, which is usually the most serious type of whiplash injury [28].

Whiplash injury has been shown to damage deep tissues in the facet joint by compression and/or stretching, and to damage the disc by shear forces [43]. Autopsy studies also have documented disc herniation [60], annulus-fibre ruptures [29] and cartilage-endplate separations [124] due to frontal impacts. Peak annulus fibrosus (AF) fibre strain is generally concentrated in the posterolat-

Table 4. Summary of cervical disc injury mechanisms

Mechanism	Number of discs	Threshold value		Reference
Tension		Failure load	Ultimate strength/displacement	
	7 A C4–C5 segment, and a C5–C6–C7 segment.	88 kg 2639 N	0.3 kg·mm ⁻² 3.9 mm	[119] [30]
Axial torsion	11	51 kg	0.58 kN 0.86 kN 0.48 kg·mm ⁻² 5 N·m	(F. A. [96]) (H. [136]) [119] (H. [136])
Compression	Several	320 kg	1.08 kg·mm ⁻² 3.14 kN	[119] (H. [136])
Flexion	A C4–C5 segment, and a C5–C6–C7 segment	Failure load 20.9 N·m	Failure angle (°) 13.7	[30]
Extension		22.4 N·m	19.6	
Strain energy absorption capacity	5 normal intervertebral joints and 4 degenerated intervertebral joints.	The total strain energy capacity of the system $U_{tot} < U_y$, (10.20 J \pm 1.90 J for normal intervertebral joints; 4.04 J \pm 0.83 J for degenerated intervertebral joints)		[142]

eral portion of the disc, and the highest amount of strain has been found in the C4–C5 and C5–C6 discs. The C2–C3 intervertebral level displayed greater increases during frontal impact, while during rear-end impact, C5–C6 was at the greatest risk. The findings of Ito et al.’s study suggested that injuries observed at C2–C3 and C5–C6 might be due to increases in disc annular tissue strain and disc shear strain beyond the physiological limits [55]. Both the posterior disc and the facets are compressed, causing disc contusion or herniation, facet hemarthrosis, bruising around the C2 nerve, or fractures of articular processes. Suboccipital vascular congestion and annulus calcification are also seen [28]. In addition, varying the stiffness of the interspinous ligament (ISL) was found to greatly affect the intervertebral disc pressure (IDP). The decreased stiffness of the ISL and the CL would place greater stress on the intervertebral disc, which might contribute to degeneration [55]. The threshold for intervertebral disc injury has been explored in *in vitro* experiments such as stretching, compression, axial torsion, buckling and stretching. Table 4 summarizes several representative intervertebral disc tolerance-limits experiments.

2.1.4 Other soft-tissue injury mechanisms. High-speed glass debris, broken bones and seat belts in automobile accidents can cause neck injuries, such as soft-tissue contusions or cuts, with the most lethal being carotid artery injury. The amplitude, rate and time of vertebral artery elongation also cause vertebral artery injury. The average vertebral artery elongation between the occipital and C6 vertebrae has been measured using a custom

transducer mounted on the neck of the corpse [31]. The peak vertebral artery elongation was 30.5 mm during the posterior impact on the head and 17.4 mm during the side impact, significantly exceeding the physiological elongation limit. Due to the Poisson effect, vertebral artery elongation leads to a decrease in vessel diameter and may result in transient vascular damage. Alternatively, the container can be stretched or squeezed along a turn during the twisting process [9]. These mechanisms can also cause tearing of the vertebral artery intimal layer [21]. Further biomechanical studies are needed to determine the strain distribution of the entire vertebral artery and the loading rate associated with whiplash during physiological movements with different initial neck postures and various impact directions.

2.2 Neck fracture mechanisms

The cervical spine contains seven pieces, among which C1 and C2 are two unique pieces. C1 is referred to as the atlas and is ring-like. C2 has an obvious dens that can work together with C1. The neck injury mode depends on the load on the neck, which can be divided into compression of the neck, tension, shear, flexion moment, extension moment and axial torsion [79].

Shear and axial torsion in the anterior-posterior direction may result in dislocation of the atlanto-occipital joint, while large compression may result in a fracture of the atlas (a Jefferson fracture). If axial compression is combined with neck extension, a C2 fracture may occur, commonly referred to as a hangman’s fracture. In car

accidents, this type of fracture is usually associated with impact on the forehead or face, such as from the windshield or the steering wheel [130]. The interaction between the head and the roof structure may cause neck injuries when the occupant is turned upside down during rollover [101]. Damage caused by tensile load includes dislocation of the occipital condyles, ligamentous injury and fractures [88]. Bending is also an important cause of cervical spine injuries. Its forms of force include compression-flexion, compression-extension, tension-flexion, tension-extension and lateral bending. The combination of post-bending load and compression force can cause fractures in the back of the neck, including the upper and lower regions [91, 97]. The combination of the bending moment of the neck and the axial compression force can cause a wedge fracture of the anterior vertebral body [79]. With the increase of force, there may be a burst fracture or bilateral facet dislocation. The latter two conditions are unstable and may disrupt or injure the spinal cord. The extent of the injury in such cases depends on the penetration of the vertebral body or its fragments into the spinal canal [130]. When the human body is restrained during a collision, the cervical vertebrae are subjected to tensile force and bending moment due to inertia. Bilateral facet dislocation has been observed after such loading [79]. When the chin collides with a hard object, tension-extension loading is a potential cause of damage. At this time, C2 may suffer a hangman's fracture, and the soft tissue of the neck may also be damaged. During side impacts, lateral bending can also cause damage to the cervical spine, which usually occurs at the same time as compression or shearing. The combination of lateral bending and compression can cause a fracture of the cervical vertebra on the compression side. The combination of lateral bending and torsion results in unilateral facet dislocations or unilateral locked facets [83].

Large numbers of cadaveric experiments and tests involving volunteers have been conducted to measure the tolerance of cervical spine injury. Volunteer experiments, usually performed under low-load conditions, provide only pain feedback from volunteers. It is difficult to determine the load strength of a vertebral fracture. Cadaveric experiments usually use either the entire cervical vertebra or a segment to simulate the movement of the neck (stretching, compressing, bending and stretching) during a car accident. Table 5 summarizes the damage mechanisms and tolerance lim-

its of neck fractures examined in various studies. Some of these studies, especially the volunteer experiments, did not specify the details of the neck injuries examined.

2.3 Cervical spinal cord injury mechanisms

The spinal cord runs within the vertebral canal formed by the back parts of the vertebrae. Thirty-one pairs of nerves branch out from the spinal cord through the vertebrae, carrying messages between the brain and every other part of the body. SCI occurs when a fracture, dislocation or other neck injury damages the spinal cord. People living with SCI often endure a lifelong disability with complete or incomplete paralysis below the level of injury. If the spinal cord is damaged at the third cervical vertebra or above, the person may die or need a respirator to stay alive.

Clinical evidence suggests a relationship between column injury patterns (such as burst fracture, fracture dislocation, transverse contusion or distraction) and spinal cord injury [45]. This relationship may be due to the mechanical properties of the column-damage pattern, which can cause a certain degree of spatial distribution and damage to the spinal cord tissue [45]. By analysing patients with cervical spine injuries, Kiwerski concluded that the most common causes of injury were the flexion (48%), compressive (26%) and hyperextension (26%) mechanisms [66].

SCI is closely related to the values of mechanical strain and stress which the spinal cord are subjected to during trauma [8, 75]. In the study by Czyz et al., based on statistical analysis, the highest values of stress and strain were found in the anterior spinothalamic, lateral spinothalamic and dorsal column tracts at the time of trauma [26]. In Ghaemi and Bahramshahi's study, two models were established and analysed for comparison to study spinal cord behaviour under various loading conditions [42]. The pair found that in compression, there was more stress, strain and displacement on the anterior surface of the spinal cord than on the posterior surface. In flexion and extension, the posterior surface of the spinal cord saw more displacement, stress and strain than the anterior. They found that stress on the spinal cord was higher than any other loading condition in flexion. Czyz et al. designed an experiment in which 28 patients recovering from cervical spine injury were enrolled: 14 with neurological symptoms of TSCI (the study group) and 14 who were neurologically intact (the control group). Czyz et al. found that there was no

Table 5. Summary of neck fracture injury mechanisms

Mechanism	Objects	Threshold criterion	Threshold value	Reference
Extension	Volunteers	No-injury (static)	23.7 N·m	[44]
		Pain	47.3 N·m	[81]
	Cadavers	No-injury	47.5 N·m	[44]
		AIS 2	56.7 N·m	
		Upper cervical spine	49.5 (17.6 SD) N·m and 42.4° (8.0° SD)	[89]
Flexion	Volunteers	Pain	59.4 N·m	[81]
			59.7 N·m	[44]
		Maximum voluntary loading	87.8 N·m	[81]
	Cadavers	AIS 2	88.1 N·m	[44]
		Fractures	189 N·m	[81]
		Upper cervical spine	190 N·m	[44]
Tension	Volunteers	No-injury (static)	39.0 (6.3° SD) N·m and 58.7° (5.1° SD)	[89]
Compression	Cadavers	Failure	1.1 kN	[81]
	Cadavers	Bilateral facet dislocation	3.1 kN	[112]
		Compression injuries	1.72 kN	[90]
		Compression injuries	4.8–5.9 kN	[76]
		Neck injury	2.75–3.44 kN	[86]
Shear (a-p)	Volunteers	No-injury	2.4–5.3 kN	[112]
		Failure	2.243 ± 0.572 kN (male)	[91]
	Cadavers		1.061 ± 0.273 kN (female)	
			3.81 ± 0.97 kN (male)	(Frank A. Pintar [97])
			2.30 ± 1.10 kN (female)	
Functional unit	Volunteers	No-injury	845 N	[81]
	Cadavers	Irreversible damage	2 kN	[44]
	Functional unit	Odontoid fractures	1.5 kN	[32]

relation between age, gender and level of injury, and levels of strain and stress [27]. In addition, after SCI, there were some risk factors of neurological deficit, including the results in the longitudinal axis (z), in stress and strain. The cut-off value for stress was 8.1 kPa, and for strain 0.0117. The results for stress and strain correspond with grading on the ASIA scale. One grade change on the ASIA scale is associated with a decrease on the z-axis of 4.01 kPa and 0.012 for stress and strain, respectively. Czyz et al. found that the severity of osseous and ligament structural damage has a significant effect on the mechanical stress range of the spinal cord. Spinal nerve tissue is most resistant to mechanical forces acting in the sagittal direction, and distraction is most destructive to SCI [27]. In Greaves's study, three models of a three-vertebrae segment of the human cervical spine and spinal cord were developed and validated in order to simulate three injury mechanisms, including transverse contusion, distraction and dislocation. The study found that the contusion and dislocation mechanisms resulted in higher strain gradients near the damage site, while the distraction mechanism produced a more uniform strain distribution on the width and length of the spinal cord. The

study highlighted the greater possibility of grey matter damage due to mechanical or biological susceptibility than of damage to the white matter.

High stress and high strain on the spinal cord are related to the impact strength of car accidents (Table 6). Khuyagbaatar, Kim and Kim found that when the initial impact velocity exceeded the threshold of 4.5 m/s, spinal cord stress increased significantly as the velocity of the test pellet increased, regardless of the pellet size, resulting in increased cord compression, cross-sectional area reduction and obliteration of the cerebrospinal fluid [62]. It has been demonstrated that during a deeper impact, contusion is more sensitive to impact velocity than during a shallow impact. Bone fragment impact on the spinal cord considered to be another parameter of SCI. Bone fragments of vertebral bodies with high impact velocity cause SCI and various degrees of neurological deficits. In animal tests, impact velocity has shown an interaction with impact depth that becomes more important in determining the severity of the damage beyond the depth threshold [69]. An overall increase in spinal cord deformation results from an increase in impact velocity [46]. A fragment produced by a burst fracture moving at

Table 6. Summary of cervical spinal cord injury mechanisms

Mechanism	Object	Methodology	Threshold value	Reference
Head impact velocity	Adult	Accident investigation	> 3.1 m/s	[80]
Fragment impact velocity	Animal (bovine)	Simulation (FE model)	> 4.5 m/s	[62]
Stress	Adult	Simulation (FE model)	> 8.1 kPa	[27]
Strain	Animal (rat)	Simulation (FE model)	> 0.0117	
			> 0.1 maximum principal strain corresponded to elevated average levels of tissue damage	[104]
Compression force	Animal (minipig)	Animal tests	< 1.5 kg without injury or able to recover after injury	[87]
			> 2.5 kg with injury and unable to automatically recover	
Multiple	Adult	Accident investigation	Flexion mechanism is main reason for injury (accounts for 48%)	[66]
			Most serious sequela is observed in crush fractures of vertebrae (76% of patients)	
Compression and flexion	Animal (bovine)	Simulation (FE model)	Stress distribution of spinal cord under static compression increased with greater flexion speed of spinal cord	[61]
Material properties	Animal (bovine)	Animal tests	Grey matter is more rigid and fragile than white matter	[53]
	Animal (rat)		Lower injury threshold of the highly vascularized grey matter in comparison to white matter	[75]
	Adult	Simulation (FE model)	Preferential damage to grey matter in comparison to white matter	[45]

a high velocity may have a major impact on spinal cord deformation and will maximize stress on the spinal cord. Sparrey et al. reported that high-speed injury caused the axons around the centre of the spinal cord to be severed immediately, resulting in increased dorsal/ventral haemorrhage [120].

2.4 Neck injury criteria

In order to quantify the relationship between mechanical load and human neck injury risk, many quantitative criteria for neck injury have been proposed based on various damage mechanisms for different load environments. Common neck injury indicators include the Neck Injury Criterion (NIC), the N_{ij} injury criterion, the N_{km} injury criterion, the Lower-Neck Load Index (LNL) and the Neck Displacement Criterion (NDC). These guideline criteria are a basis for judging the design of neck safety devices and safety strategies.

The NIC predicts the relationship between spinal cord nerve tissue damage and pressure gradients. Boström et al. assumed that pressure gradients caused by a sudden change of the fluid flow inside the fluid compartments of the cervical spine are related to neck injuries [14]. Based on

the results of animal experiments, Boström et al. simulated the neck injury index of the spinal canal pressure pulse injury mechanism and defined the NIC as follows:

$$NIC(t) = 0.2a_{rel}(t) + v_{rel}(t)^2 \quad (1)$$

where a_{rel} and v_{rel} are the relative horizontal acceleration and velocity between the bottom (T1) and top (C1) of the cervical spine. The constant, 0.2, represents the approximate length of the neck in metres. This equation accounts for what is now widely held to be one of the most important risk factors in Low Speed Rear Impact Collision Cases (LOSRIC) injury—the retraction of the head (head lag) during the first 100 ms or so of the crash sequence [15, 115]. The damage threshold is 15. When the NIC was below 8, the volunteers in Boström et al.'s study did not experience any symptoms; when the NIC was 10, some volunteers reported pain. In the cadaver experiment, when the NIC was 18.6, ligament tears occurred.

The N_{ij} neck injury criterion was proposed by the US National Highway Traffic Safety Administration (NHTSA) to assess severe neck injuries (AIS2+) from frontal impacts, including those with

airbag deployment and thus accounting for more severe impact conditions at higher Δv [67]. The N_{ij} criterion implies a linear combination of the axial forces and the flexion/extension bending moment, both normalized by critical intercept values:

$$N_{ij} = \frac{F_z}{F_{int}} + \frac{M_y}{M_{int}} \quad (2)$$

where F_z and M_y are the axial force and the sagittal bending moment, respectively. F_{int} and M_{int} indicate the critical force and critical intercept values. The threshold for damage is N_{ij} equal to 1. For the Hybrid III mechanical dummy (for frontal impact), the critical values of axial force and bending moment are as follows: F_{int} (tension) = F_{int} (compression) = 4500 N; M_{int} (flexion) = 310 N·m; and M_{int} (extension) = 125 N·m.

Since the critical values are proposed based on a specific mechanical dummy, it is necessary to redefine the critical values of each load when measuring the N_{ij} value with different mechanical dummies. The N_{ij} is used mainly to study neck injury in frontal-impact vehicle accidents.

The N_{km} was proposed by Schmitt et al. [108]. It is based on a linear combination of shear forces acting in the sagittal direction and extension/flexion bending moments, both measured at the occipital condyles. A similar approach led to the definition of the N_{ij} criterion for frontal impact, and thus the newly proposed N_{km} can be regarded as a modification of the former. The N_{km} criterion was defined according to the following equation:

$$N_{km} = \frac{F_x(t)}{F_{int}} + \frac{M_y(t)}{M_{int}} \quad (3)$$

where $F_x(t)$ and $M_y(t)$ are the shear force and the flexion/extension bending moment, respectively; both values should be obtained from the load cell positioned at the upper neck. F_{int} and M_{int} represent critical intercept values used for normalization. N_{km} is used primarily to study neck injury in rear-end-impact vehicle accidents. The threshold of N_{km} is 1. For the Hybrid III mechanical dummy, the critical values of shear force and bending moment are: F_{int} (forward) = F_{int} (backward) = 4500 N; M_{int} (flexion) = 310 N·m; and M_{int} (extension) = 125 N·m.

The LNL is an indicator of the risk of neck injury based on the load at the T1 thoracic vertebra. This indicator is very sensitive to the design parameters of the safety seat and is consistent with the mechanism of facet joint injury supported by Yoganandan

et al. [141]. Its calculation formula is as follows:

$$LNL = \frac{|M_{ylw}|}{M_c} + \frac{|F_{xlw}|}{F_{xc}} + \frac{|F_{zlw}|}{F_{zc}} \quad (4)$$

where M_{ylw} , F_{xlw} and F_{zlw} are the bending moment, shear force and axial force measured at the T1 thoracic vertebra, and the reference values are 15 N·m, 250 N and 900 N, respectively.

From their analysis of the kinematics of volunteers, Viano and Davidsson developed the NDC [132]. The angular velocity and line displacement of the head relative to the T1 thoracic vertebra were used as indicators of neck injury. The NDC was given by the threshold channel on two graphs. The horizontal axis was the vertical displacement and rotation angle of the skull occipital sac relative to the T1 thoracic vertebra, and the vertical axis was the horizontal posterior displacement of the skull occipital sac. In their figures, the threshold channel divided the experimentally measured head and neck dynamics into four grades: excellent, good, qualified and unqualified.

The Intervertebral Neck Injury Criterion (IV-NIC) is defined as the ratio of the intervertebral motion under traumatic loading and the physiological range of motion [92]. It is impossible to evaluate dummies using the IV-NIC, as dummies are not capable of simulating intervertebral motion. In addition, the criterion is neither validated, nor is there a threshold level proposed.

3. Prevention of neck injury

3.1 Passive safety

The main principle of safety devices that protect the neck is to reduce the inertia generated by the relative movement of the head and the body. Based on this principle, the softened seat back, the active head restraint, the smart head restraint, the energy-absorbing seat base, the seat belt, the airbag and other devices have been developed for the protection of the vehicle occupant's head and neck. With advancements in the understanding of neck injuries in vehicle accidents and the development of simulation/experimentation techniques, designs for neck protection devices are becoming increasingly scientific, integrated and intelligent.

These devices have been developed step by step along with advancements in the understanding of the mechanism of neck injuries. It was initially thought that hyperextension of the neck was the main cause of neck injury in a vehicle accident,

as a result of which head restraints, rear shifting backrests and seat energy-absorbing devices were developed. A developed mathematical approach was established via the simulation of human neck behaviour using the global seat back model and the advanced global seat back model [117]. Song et al. used this approach to analyse four different design parameters, including head-to-headrest distance, seat-back joint stiffness, upper seat-back stiffness and lower seat-back stiffness [117]. They drew the conclusion that softening the seat back could improve headrest performance and increase the head-torso extension angle and the moment force at the C7/T1 joint. The Volvo Whiplash Protection Study (WHIPS) was designed to control the movement of the backrest in a rear-end impact [72]. The WHIPS seat also reduced forward rebound and gave improved closeness, as well as improving distributed load support of the back and head on the basis of tests. Building on the WHIPS research, Jakobsson, Isaksson-Hellman and Lindman demonstrated that the WHIPS seat supported the head and avoided hyperextension of the neck in a rear-end impact by analysing experimental data for vehicle occupants [59]. Schmitt et al. used two steel profiles as energy-absorbing elements that plastically deformed and absorbed energy as the seat moved backwards [107]. This device reduced the maximum NIC and the relative displacement between the head and T1, as well as T1 acceleration. A seat slide mechanism was designed by Luo and Zhou through quasi-static tests and finite-element simulations. A MADYMO numerical model consisting of a BioRID-II rear-impact dummy, seat and vehicle floor was established, and a quasi-static test demonstrated the process by which the seat absorbed energy through deformation [73]. Based on Luo and Zhou's research, Zhang and Zhou developed a sliding seat prototype based on the bending of a steel trip, and used a sled test to verify its structural effectiveness [145]. Hassan and Meguid used the Global Human Body Model Consortium (GHBMC) finite-element model to study the effect of seat belt use on injury to the vehicle occupant's neck during rear-end impact [49]. Results obtained by comparative experiments showed that the seat belt was capable of protecting the body from vertical ramping during rear-end impact and ensuring the correct position of the headrest and the head. In addition, the seat belt was able to prevent the occupant from moving forward uncontrolled, and colliding with the steering wheel or the windshield.

The headrest reduces the extent of neck damage. However, whiplash in traffic accidents persists. Volunteer low-speed collision studies have shown that whiplash may be associated with early neck movement during the collision, and that hyperextension theory does not correctly explain whiplash. In order to reduce the bending amplitude of the neck in the early stage of the collision process, active headrests and smart headrests have been developed. Saab were the first to introduce an active head restraint on the Saab 9-3 [135]. This was a mechanical structure that utilized leverage to implement protection of the neck during rear-end impact. When a rear-end collision occurred, the force pressing the seat back from the occupant could be converted into forward and upward movement of the head restraint. This action limited the movement of the head so that the cervical ligament did not bend excessively. The Neck-Pro released by Mercedes-Benz was the first proactive head restraint [3]. When the collision signal received by the sensors reached a preset minimum value, the electronic system directed the active head restraint to release the previously compressed spring to eject the head restraint, thereby reducing the backset and topset. Loughborough University invented a head restraint called the Smart Head Restraint in 2007. The device used a pair of ultrasonic sensors to detect the position of the head and direct the head restraint to automatically move to the most suitable position by control algorithms and mechanical structures. It was expected to protect the neck effectively during the driving process [3]. A concept for an integrated whiplash-mitigating head restraint and seat has also been proposed [2]. The concept combines the functions of the reaction head restraint and Volvo's WHIPS system, to produce a seat recliner that moves with the head restraint simultaneously during a rear-end collision. It has been found that the device leads to a greater degree of protection of the neck.

3.2 Active safety and neck injury prevention for intelligent vehicles

Passive safety systems have advanced considerably over the years, and the automotive industry has shifted its attention to active safety, where there are still a great many new unexplored areas. Active safety is increasingly being used to describe systems that use an understanding of the state of the vehicle to avoid or minimize the effects of a crash. These advanced driver-

assistance systems (ADAS) include braking systems, such as brake assist, traction control systems and electronic stability control systems, which interpret signals from various sensors to help the driver control the vehicle. Active safety research today focuses primarily on sensor-based systems, such as advanced driver-assistance systems including adaptive cruise control and collision-warning/avoidance/mitigation systems.

Collision-avoidance systems use radar (all-weather) and sometimes lasers (LIDAR) and cameras (employing image recognition) to detect an imminent crash. Auto Emergency Braking (AEB) is a feature that alerts drivers to imminent crashes and helps them use the maximum braking capacity of the car. AEB will independently brake if the situation becomes critical and no human response is registered. Yamada et al. studied the way in which AEB could reduce injury by reducing the maximum forward displacement of vehicle occupants [137]. Also due to its warning sound emitted before its activation, it has been proved that AEB reduced the head acceleration compared to a human break [105]. In addition to acting alone, ABS can also reduce the impact on occupants in collisions in conjunction with other active safety technologies such as the pre-safety seatbelt (PSB). When AEB is working, the appropriate activation time of the PSB can reduce head and neck injuries [63]. In a study of police-reported crashes, AEB was found to reduce the incidence of rear-end crashes by 39% [22]. Rizzi, Kullgren and Tingvall calculated the overall benefits of AEB, in terms of both crash avoidance and injury mitigation, finding that the reduction of striking rear-end crashes in 50 km/h speed areas ranged between 54% and 57% [102].

In addition to AEB, Forward Collision Warning (FCW) can also reduce rear-end crashes [22]. There are also other active safety technologies, such as Forward Collision Warning and Brake support combined with Adaptive Cruise Control (CWB+ACC) and the Advanced Automatic Crash Notification system (AACN), that have a significant impact on the collision safety of cars. For vehicles with CWB+ACC, rear-end frontal collisions are reduced by 38% [54]. Bose et al. developed a computational methodology to enhance the existing AACN framework, and found a significant improvement in post-crash injury prediction [12].

In future, highly automated vehicles, especially driverless ones, pose new challenges to passenger crash safety. In highly automated vehicles, the posture of the occupant is complex and changeable—the posture of the driver in particular has changed

significantly—and different postures produce different injuries in the event of collision. Shateri and Cronin demonstrated that the location of injury or pain depended on the occupant's initial posture, and that head position therefore had considerable influence when a crash occurred [111]. Hault-Dubrulle et al. investigated the effect of pre-collision occupant posture on injury outcome during collisions—with three typical responses being bracing rearward into the seat, straightening the arms against the steering wheel and swerving in an attempt to avoid the impacting vehicle—and found that an out-of-position (OOP) situation has a huge influence on injury [50]. Bose et al. found occupant posture to be the most significant parameter affecting the overall risk of injury in frontal collisions, out of the four parameters of mass, stature, posture and bracing level [13]. Passenger posture is therefore an important issue for passenger crash safety in driverless cars.

Changes in the occupant's posture raises the requirements for a restraint system. In a highly automated vehicle, the driver's head may be in a non-neutral position, which may increase the potential for injury during a crash [4]. One possible occupant position in a self-driving car is leaning against the seat back while the seat back is reclined. As the seat back recline angle increases, the belt fit worsens and may cause greater injury to the occupants when a crash occurs. Alternatively, occupants of highly automated vehicles may not be belted or restrained in their seats, which may cause greater injury when crashes occur. In the event of a crash, the impact time of the occupant's head and the airbag is also different for OOP occupants, which leads to significant differences in dummy head accelerations [99]. The effect of a reclined seat back in a moving vehicle on the deterioration of occupant protection and modification of the injury pattern was the subject of Thorbole's study; the results showed that the effect of the belt was diminished when a crash occurred with a reclined seat back [125]. Even on autonomous vehicles, we then do not recommend to letting the possibility to recline too much the seatback, as well as to not wear seatbelts.

The adaptive restraint system brings opportunities to solve the problem of the self-driving vehicle occupant's posture. The adaptive restraint system automatically adjusts the characteristics of the occupant-restraint system based on the strength of the collision and the characteristics of the occupant at the time of the accident, thereby minimizing the risk of occupant injury after a col-

lision. The working principle of the system is that when the sensor registers a collision, the system will automatically adjust the airbag detonation [7], the pre-tension of the seat belt [63] and the position of the headrest [64, 65, 94] according to the location and the severity of the collision, which can minimize the relative displacement between head and torso when collision, especially rear-end collision, occurs. This solves the problem of occupant posture, especially the optimal matching relationship between the spatial position of the head and neck and the restraint system. Compared to traditional restraint systems, the adaptive restraint system incorporates active headrests and can more effectively reduce the risk of neck injury to the occupant in the event of rear-end collisions [4, 52, 126]; moreover, the addition of the decision-making system enables more precise control of the initiation of the airbag and the preload of the seat belt in collisions [13]. The risk of occupant injury is further reduced, and the adaptability of the entire system is more efficient and more stable. In the field of smart cars, by cooperating with the sensor system of the vehicle, the adaptive restraint system can predict the position where the collision will occur, and automatically calibrate the occupant-restraint system according to the conditions of the occupant's sitting posture. The parameters are adjusted so that the probability of the occupant's being injured in the event of a collision is greatly reduced, as is the safety risk caused by the occupant's being OOP.

Beside the posture issue, the level of attention will be different, or even non-existent, in future autonomous vehicles. Mainly due to the fact that the rules on the roads will be respected, and if an event happens, it will be at lower energy than nowadays [123]. This is why research in this field has to focus both on numerical simulation and in vivo tests. To access the internal body injury mechanisms (numerical models) on one side, and to continue to validate the models, at low acceleration levels, taking into account the physiological aspects of the living human body (experimental tests), on the other side.

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References

1. Abu Naser SS, Almursheidi SH. A knowledge based system for neck pain diagnosis. *W W J Multidiscip Res Dev* 2016; 2:12–8.
2. Acar M, Bewsher SR. Design concepts for an integrated whiplash mitigating head restraint and seat. *Int J Crashworthiness* 2016; 21:79–88.
3. Acar M, Clark SJ, Crouch R. Smart head restraint system. *Int J Crashworthiness* 2007; 12:429–35.
4. Adam T, Untaroiu CD. Identification of occupant posture using a Bayesian classification methodology to reduce the risk of injury in a collision. *Transport Res C-Emer* 2011; 19:1078–94.
5. Adams MA, Hutton WC. Prolapsed intervertebral disc: a hyperflexion injury. *Spine* 1982; 7:184–91.
6. Adams M, Hutton W. The effect of fatigue on the lumbar intervertebral disc. *J Bone Joint Surg Br* 1983; 65-B: 199–203.
7. Bai Z, Jiang B, Zhu F, et al. Optimizing the passenger air bag of an adaptive restraint system for multiple size occupants. *Traffic Inj Prev* 2014; 15:556–63.
8. Bain AC, Meaney DF. Tissue-level thresholds for axonal damage in an experimental model of central nervous system white matter injury. *J Biomech Eng* 2000; 122:615–22.
9. Barton JW, Margolis MT. Rotational obstruction of the vertebral artery at the atlantoaxial joint. *Neuroradiology* 1975; 9:117–20.
10. Berglund A, Alfredsson L, Jensen I, et al. Occupant- and crash-related factors associated with the risk of whiplash injury. *Ann Epidemiol* 2003; 13:66–72.
11. Biering-Sørensen F, Bickenbach JE, El Masry WS, et al. ISCoS-WHO collaboration. International Perspectives of Spinal Cord Injury (IPSCI) report. *Spinal Cord* 2011; 49:679–83.
12. Bose D, Crandall JR, McGwin G, et al. Computational methodology to predict injury risk for motor vehicle crash victims: a framework for improving Advanced Automatic Crash Notification systems. *Transport Res C-Emer* 2011; 19:1048–59.
13. Bose D, Crandall JR, Untaroiu CD, et al. Influence of pre-collision occupant parameters on injury outcome in a frontal collision. *Accident Anal Prev* 2010; 42:1398–407.
14. Boström O, Svensson MY, Aldman B, et al. A new neck injury criterion candidate-based on injury findings in the cervical spinal ganglia after experimental neck extension trauma. In: *Proceedings of the 1996 International Ircobi Conference on the Biomechanics of Impact*. Ireland: Dublin, 1996, 123–36.
15. Brault JR, Wheeler JB, Siegmund GP, et al. Clinical response of human subjects to rear-end automobile collisions. *Arch Phys Med Rehab* 1998; 79:72–80.
16. Brault JR, Siegmund GP, Wheeler JB. Cervical muscle response during whiplash: evidence of a lengthening muscle contraction. *Clin Biomech* 2000; 15:426–35.
17. Buonocore E, Hartman JT, Nelson CL. Cineradiograms of cervical spine in diagnosis of soft-tissue injuries. *JAMA-J Am Med Assoc* 1966; 198:25–9.
18. Carroll LJ, Holm LW, Hogg-Johnson S, et al. (2009). Course and prognostic factors for neck pain in whiplash-associated disorders (WAD): results of the Bone and Joint Decade 2000–2010 Task Force on Neck Pain and Its Associated Disorders. *J Manip Physiol Ther* 2009; 32:S97–107.
19. Cassidy J, Spitzer W, Skovron M, et al. The Quebec Task Force on Whiplash-Associated Disorders. *Spine* 1996; 21:897–8.
20. Chen H, Yang KH, Wang Z. Biomechanics of whiplash injury. *Chin J Traumatol* 2009; 12:305–14.
21. Chung Y, Han D. Vertebrobasilar dissection: a possible role of whiplash injury in its pathogenesis. *Neurol Res* 2002; 24:129–38.
22. Cicchino JB. Effectiveness of forward collision warning systems with and without autonomous emergency braking in reducing police-reported crash rates. In: *Technical report*. Insurance Institute for Highway Safety, 2016.

23. Curatolo M, Bogduk N, Ivancic PC, et al. The role of tissue damage in whiplash-associated disorders. *Spine* 2011; **36**:S309–15.
24. Cusick JF, Yoganandan N. Biomechanics of the cervical spine 4: Major injuries. *Clin Biomech* 2002; **17**:1–20.
25. Cusick J, Yoganandan N (2001). Whiplash Syndrome Kinematic Factors Influencing Pain Patterns.pdf, (July).
26. Czyz M, Ścigała K, Bedziński R, et al. Finite element modelling of the cervical spinal cord injury-clinical assessment. *Acta Bioeng Biomech* 2012; **14**:23–9.
27. Czyz M, Tykocki T, Miękisiak G, et al. Critical values of mechanical stress and strain during traumatic cervical spinal cord injury: clinical study with the use of Finite Element Modelling. *J Biomed Graph Comput* 2016; **6**:22–30.
28. Davis CG. Mechanisms of chronic pain from whiplash injury. *J Forensic Leg Med* 2013; **20**:74–85.
29. Davis SJ, Teresi LM, Bradley WG, et al. Cervical spine hyperextension injuries: MR findings. *Radiology* 1991; **180**:245–51.
30. JA DW, Cronin DS. Cervical spine segment finite element model for traumatic injury prediction. *J Mech Behav Biomed* 2012; **10**:138–50.
31. Dobrin PB. Mechanical properties of arteries. *Physiol Rev* 1978; **58**:397–460.
32. Doherty BJ, Heggeness MH, Esses SI. A biomechanical study of odontoid fractures and fracture fixation. *Spine* 1993; **18**:178–84.
33. Dreyer SJ, Boden SD. Natural history of rheumatoid arthritis of the cervical spine. *Clin Orthop Relat R* 1999; **366**:98–106.
34. Dvorak J, Schneider E, Saldinger P, et al. Biomechanics of the craniocervical region: the alar and transverse ligaments. *J Orthop Res* 1988; **6**:452–61.
35. Easter JS, Barkin R, Rosen CL, et al. Cervical spine injuries in children, part II: management and special considerations. *J Emerg Med* 2011; **41**:252–6.
36. Farfan HF, Cossette JW, Robertson GH, et al. The effects of torsion on the lumbar intervertebral joints: the role of torsion in the production of disc degeneration. *Journal of Bone and Joint Surgery* 1970; **52**:468–97.
37. Feng H, Ning G, Feng S, et al. Epidemiological profile of 239 traumatic spinal cord injury cases over a period of 12 years in Tianjin, China. *J Spinal Cord Med* 2011; **34**:388–94.
38. Fice JB, Cronin DS, Panzer MB. Cervical spine model to predict capsular ligament response in rear impact. *Ann Biomed Eng* 2011; **39**:2152–62.
39. Fielding JW, GVB C, Lawsing JF III, et al. Tears of the transverse ligament of the atlas: a clinical and biomechanical study. *J Bone Joint Surg* 1974; **56**:1683–91.
40. Gales N, Kunz SN, Rocksén D, et al. Muscle pathologies after cervical spine distortion-like exposure: a porcine model. *Traffic Inj Prev* 2013; **14**:828–34.
41. Garrett, William E. Muscle strain injuries. *The American Journal of Sports Medicine*, 1996; **24**: doi:10.1177/036354659602406S02.
42. Ghaemi H, Bahramshahi N. Evaluation of the effect of cervical spine compression and sagittal moments on the spinal cord using finite element method. *Int J Biomed Eng Technol* 2012; **9**:260–76.
43. Goel VK, Monroe BT, Gilbertson LG, et al. Interlaminar shear stresses and laminae separation in a disc: finite element analysis of the L3-L4 motion segment subjected to axial compressive loads. *Spine* 1995; **20**:689–98.
44. Goldsmith W, Ommaya AK. Head and neck injury criteria and tolerance levels. *The Biomechanics of Impact Trauma*. 1984: 149–87.
45. Greaves CY, Gadala MS, Oxland TR. A three-dimensional finite element model of the cervical spine with spinal cord: an investigation of three injury mechanisms. *Ann Biomed Eng* 2008; **36**:396–405.
46. Hall RM, Oakland RJ, Wilcox RK, et al. Spinal cord-fragment interactions following burst fracture: an in vitro model. *J Neurosurg Spine* 2006; **5**:243–50.
47. Harvey L. *Management of Spinal Cord Injuries E-Book: A Guide for Physiotherapists*. Edinburgh: Butterworth-Heinemann, 2008
48. Hasharoni A, Errico TJ. Transverse ligament injury. In: Clark CR (ed). *The Cervical Spine* 4th edn. Philadelphia, PA: Lippincott Williams & Wilkins, 2005, 608–13
49. MTZ H, Meguid SA. Effect of seat belt and head restraint on occupant's response during rear-end collision. *Int J Mech Mater Des* 2018; **14**:231–42.
50. Hault-Dubrule A, Robache F, Delille R, et al. Influence of pre-crash driver posture on injury outcome: airbag interaction with human upper extremities. *Comput Methods Biomech Biomed Eng* 2012; **15**:295–7.
51. Hein C, Richter H, Rath SA. Atlantoaxial screw fixation for the treatment of isolated and combined unstable Jefferson fractures - experiences with 8 patients. *Acta Neurochir* 2002; **144**:1187–92.
52. Hynd D, Carroll J, Cuerden RW, et al. Restraint system safety diversity in frontal impact accidents. In: *Proceedings of the International Research Council on the Biomechanics of Injury Conference*. Ireland: Dublin, 2012, 114–29
53. Ichihara K, Taguchi T, Shimada Y, et al. Gray matter of the bovine cervical spinal cord is mechanically more rigid and fragile than the white matter. *J Neurotraum* 2001; **18**:361–7.
54. Isaksson-Hellman I, Lindman M. Evaluation of rear-end collision avoidance technologies based on real world crash data. In: *Proceedings of the 3rd International Symposium on Future Active Safety Technology Towards Zero Traffic Accidents*. Sweden: Gothenburg, 2015, 471–6
55. Ito S, Ivancic PC, Pearson AM, et al. Cervical intervertebral disc injury during simulated frontal impact. *Eur Spine J* 2005; **14**:356–65.
56. Ivancic PC, Panjabi MM, Tominaga Y, et al. Spinal canal narrowing during simulated frontal impact. *Eur Spine J* 2006; **15**:891–901.
57. Ivancic PC, Pearson AM, Panjabi MM, et al. Injury of the anterior longitudinal ligament during whiplash simulation. *Eur Spine J* 2004; **13**:61–8.
58. Ivancic PC, Xiao M. Understanding whiplash injury and prevention mechanisms using a human model of the neck. *Accident Anal Prev* 2011; **43**:1392–9.
59. Jakobsson L, Isaksson-Hellman I, Lindman M. WHIPS (Volvo cars' Whiplash Protection System) - the development and real-world performance. *Traffic Inj Prev* 2008; **9**:600–5.
60. Jónsson H, Bring G, Rauschnig W, et al. Hidden cervical spine injuries in traffic accident victims with skull fractures. *J Spinal Disord* 1991; **4**:251–63.
61. Kato Y, Kanchiku T, Imajo Y, et al. Flexion model simulating spinal cord injury without radiographic abnormality in patients with ossification of the longitudinal ligament: the influence of flexion speed on the cervical spine. *J Spinal Cord Med* 2009; **32**:555–9.
62. Khuyagbaatar B, Kim K, Kim YH. Effect of bone fragment impact velocity on biomechanical parameters related to spinal cord injury: a finite element study. *J Biomech* 2014; **47**:2820–5.

63. Kim ES, Min SK, Sung DH, et al. The AEB system with active and passive safety integration for reducing occupants' injuries in high-velocity region. In: *24th International Technical Conference on the Enhanced Safety of Vehicles*, Gothenburg, Sweden, 2015; 15-0335.
64. Kistipadu R. Effect of reactive car seats and active head restraint system in rear-end collision and safety mechanisms to reduce whiplash injuries. Ph.D. Thesis. Wichita State University 2017.
65. Kitagawa Y, Yasuki T, Hasegawa J. Research study on neck injury lessening with active head restraint using human body FE model. *Traffic Inj Prev* 2008; **9**:574-82.
66. Kiwerski J. The influence of the mechanism of cervical spine injury on the degree of the spinal cord lesion. *Paraplegia* 1991; **29**:531-6.
67. Klinich KD, Saul RA, Auguste G, et al. *Techniques for developing child dummy protection reference values. Technical report.* National Highway Traffic Safety Administration 1996.
68. Kontautas E, Ambrozaitis KV, Kalesinskas RJ, et al. Management of acute traumatic atlas fractures. *J Spinal Disord Tech* 2005; **18**:402-5.
69. Lam CJ, Assinck P, Liu J, et al. Impact depth and the interaction with impact speed affect the severity of contusion spinal cord injury in rats. *J Neurotraum* 2014; **31**:1985-97.
70. Lee TT, Green BA, Petrin DR. Treatment of stable burst fracture of the atlas (Jefferson fracture) with rigid cervical collar. *Spine* 1998; **23**:1963-7.
71. Li F, Lu R, Hu W, et al. The influence of neck muscle activation on head and neck injuries of occupants in frontal impacts. *Appl Bionics Biomech* 2018; **7279302**.
72. Lundell B, Jakobsson L, Alfredsson B, et al. The WHIPS seat - a car seat for improved protection against neck injuries in rear-end impacts. In: *16th International Technical Conference on the Enhanced Safety of Vehicles*, Windsor, Canada, 1998, 0-8.
73. Luo M, Zhou Q. A vehicle seat design concept for reducing whiplash injury risk in low-speed rear impact. *Int J Crashworthiness* 2010; **15**:293-311.
74. MacNab I. Acceleration injuries of the cervical spine. *J Bone Joint Surg* 1964; **46**:1797-9.
75. Maikos JT, Shreiber DI. Immediate damage to the blood-spinal cord barrier due to mechanical trauma. *J Neurotraum* 2007; **24**:492-507.
76. Maiman DJ, Sances A Jr, Myklebust JB, et al. Compression injuries of the cervical spine: a biomechanical analysis. *Neurosurgery* 1983; **13**:254-60.
77. Malanga GA (ed). *Cervical Flexion-Extension/Whiplash Injuries. Spine: State of the Art Reviews.* Philadelphia, PA: Hanley & Belfus, 1998.
78. SFE M, Moulton JA, Chandrashekar N, et al. Strain rate dependent properties of younger human cervical spine ligaments. *J Mech Behav Biomed* 2012; **10**:216-26.
79. McElhaney JH, Myers BS. Biomechanical aspects of cervical trauma. In: Nahum AM, Melvin JW (eds). *Accidental Injury: Biomechanics and Prevention.* New York: Springer, 1993, 311-61.
80. JH ME, Snyder RG, States JD, et al. Biomechanical analysis of swimming pool neck injuries. *SAE Transactions* 1979; **88**:494-500.
81. Mertz HJ, Patrick LM. Strength and response of the human neck. *SAE Transactions* 1971, 2903-28.
82. Mesfar W, Moglo K. Effect of the transverse ligament rupture on the biomechanics of the cervical spine under a compressive loading. *Clin Biomech* 2013; **28**:846-52.
83. Moffatt EA, Siegel AW, Huelke DF, et al. The biomechanics of automotive cervical fractures. *Proc Am Assoc Automot Med Annu Conf* 1978; **22**:151-68.
84. Montoto-Marqués A, Ferreira-Velasco ME, Salvador-De La Barrera S, et al. Epidemiology of traumatic spinal cord injury in Galicia, Spain: trends over a 20-year period. *Spinal Cord* 2017; **55**:588-94.
85. Müller CW, Otte D, Decker S, et al. Vertebral fractures in motor vehicle accidents-a medical and technical analysis of 33,015 injured front-seat occupants. *Accident Anal Prev* 2014; **66**:15-9.
86. Myers BS, Winkelstein BA. Epidemiology, classification, mechanism, and tolerance of human cervical spine injuries. *Crit Rev Biomed Eng* 1995; **23**:5-6.
87. Navarro R, Juhas S, Keshavarzi S, et al. Chronic spinal compression model in minipigs: a systematic behavioral, qualitative, and quantitative neuropathological study. *J Neurotraum* 2012; **29**:499-513.
88. Nightingale RW, Winkelstein BA, Van Ee CA, et al. Injury mechanisms in the pediatric cervical spine during out-of-position airbag deployments. *Annu Proc Assoc Adv Automot Med* 1998; **42**:153-64.
89. Nightingale RW, Carol Chancey V, Ottaviano D, et al. Flexion and extension structural properties and strengths for male cervical spine segments. *J Biomech* 2007; **40**:535-42.
90. Nightingale RW, Doherty BJ, Myers BS, et al. The influence of end condition on human cervical spine injury mechanisms. *Stapp Car C* 1991; **35**:391-9.
91. Nightingale RW, McELHANEY JH, Camacho DL, et al. The dynamic responses of the cervical spine: buckling, end conditions, and tolerance in compressive impacts. *SAE Transactions* 1997. **106**:3968-88.
92. Panjabi MM, Wang JL, Delson N. Neck injury criterion based on intervertebral motions and its evaluation using an instrumented neck dummy. In: *Proceedings of the 1999 International IRCOBI Conference on the Biomechanics of Impact.* Spain: Sitges, 1999, 179-90.
93. Parenteau CS, Viano DC. Spinal fracture-dislocations and spinal cord injuries in motor vehicle crashes. *Traffic Inj Prev* 2014; **15**:694-700.
94. Park S, Jeong D, Kim HG, et al. New concept for neck injury lessening system development. In: *Proceedings of the FISITA 2012 World Automotive Congress.* China: Beijing, 2012, 223-36.
95. Pearson AM, Ivancic PC, Ito S, et al. Facet joint kinematics and injury mechanisms during simulated whiplash. *Spine* 2004; **29**:390-7.
96. Pintar FA. Biomechanical properties of the human intervertebral disk in tension. In: *Proceeding of 1986 ASME Adv Bioeng.*, New York, USA 1986.
97. Pintar FA, Yoganandan N, Voo L, et al. Dynamic characteristics of the human cervical spine. In: *Proceedings of the 39th Stapp Car Crash Conference.* CA, USA: San Diego, 1995, 195-202.
98. Platzer P, Jandl M, Thalhammer G, et al. Cervical spine injuries in pediatric patients. *J Trauma* 2007; **62**: 389-94.
99. Potula SR. Safety counter measures: a comprehensive crashworthiness study of out-of-position (OOP) airbag deployment and passenger impact. Ph.D. Thesis. Mississippi State University 2012.
100. Quinlan KP, Annett JL, Myers B, et al. Neck strains and sprains among motor vehicle occupants - United States, 2000. *Accident Anal Prev* 2004; **36**:21-7.

101. Ridella SA, Eigen AM. Biomechanical investigation of injury mechanisms in rollover crashes from the CIREN database. In: *Proceedings of the 2008 IRCOBI Conference*. Switzerland: Bern, 2008, 33–47.
102. Rizzi M, Kullgren A, Tingvall C. Injury crash reduction of low-speed Autonomous Emergency Braking (AEB) on passenger cars. In: *Proceedings of the 2014 IRCOBI Conference*. Berlin: Germany, 2014, 656–65.
103. Romilly DP, Skipper CS. Seat structural design choices and the effect on occupant injury potential in rear end collisions. *SAE Transactions*, 2005; **114**: 1512–20.
104. Russell CM, Choo AM, Tetzlaff W, et al. Maximum principal strain correlates with spinal cord tissue damage in contusion and dislocation injuries in the rat cervical spine. *J Neurotraum* 2012; **29**:1574–85.
105. Sandoz B, Bucsházy K, van den Berg A, et al. Acceleration of a car passenger during automatic emergency braking. In: *Proceeding of 8th World Congress of Biomechanics*, 8-12 July 2018. Dublin, Ireland.
106. Schären S, Jeanneret B. Atlasfrakturen. *Orthopade* 1999; **28**:385–93.
107. Schmitt KU, Muser MH, Heggendorf M, et al. Seat component to prevent whiplash injury. In *Proceedings of the 18th International Technical Conference on the Enhanced Safety of Vehicles*, Nagoya, Japan, 2003, 6–p.
108. Schmitt KU, Muser MH, Walz FH, et al. N km - a proposal for a neck protection criterion for low-speed rear-end impacts. *Traffic Inj Prev* 2002; **3**:117–26.
109. Schweighofer F, Stockenhuber N, Bratschitsch G, et al. Zervikale Rückenmarkverletzungen bei stabiler unterer Halswirbelsäule mit Spinalkanalstenose. *Unfallchirurg* 2002; **105**:174–7.
110. Scifert J, Totoribe K, Goel V, et al. Spinal cord mechanics during flexion and extension of the cervical spine: a finite element study. *Pain Physician* 2002; **5**:394–400.
111. Shateri H, Cronin DS. Out-of-position rear impact tissue-level investigation using detailed finite element neck model. *Traffic Inj Prev* 2015; **16**:698–708.
112. Shea M, Edwards WT, White AA, et al. Variations of stiffness and strength along the human cervical spine. *J Biomech* 1991; **24**:95–7.
113. Sidon E, Stein M, Ramalingam G, et al. Gender differences in spinal injuries: causes and location of injury. *J Women's Health* 2018; **27**:946–51.
114. Siegmund GP, Myers BS. Human cervical motion segment flexibility and facet capsular ligament strain under combined posterior shear, extension and axial compression. *Stapp Car C* 2000; **44**:159–70.
115. Siegmund GP, King DJ, Lawrence JM, Wheeler JB, Brault JR, Smith TA. Head/neck kinematic response of human subjects in low-speed rear-end collisions. *SAE transactions* 1997, Jan 1 :3877–905.
116. Siegmund GP, Winkelstein BA, Ivancic PC, et al. The anatomy and biomechanics of acute and chronic whiplash injury. *Traffic Inj Prev* 2009; **10**:101–12.
117. Song D, Uriot J, Trosseille X, et al. Modelling and analysis of interactions between occupant, seatback and headrest in rear impact. In: *Proceedings of the 1996 International IRCOBI Conference on the Biomechanics of Impact*. Ireland: Dublin, 1996, 165–85.
118. Song J, Shao J, Qi HH, et al. Risk factors for respiratory failure with tetraplegia after acute traumatic cervical spinal cord injury. *Eur Rev Med Pharmac* 2015; **19**:9–14.
119. Sonoda T. Studies on the strength for compression, tension and torsion of the human vertebral column. *J Kyoto Pref Med Univ* 1962; **71**:659–702.
120. Sparrey CJ, Choo AM, Liu J, et al. The distribution of tissue damage in the spinal cord is influenced by the contusion velocity. *Spine* 2008; **33**:812–9.
121. Sterner Y, Gerdle B. Acute and chronic whiplash disorders - a review. *J Rehabil Med* 2004; **36**:193–210.
122. Styrke J, Stålnacke BM, Bylund PO, et al. A 10-year incidence of acute whiplash injuries after road traffic crashes in a defined population in northern Sweden. *PM&R* 2012; **4**:739–47.
123. Subit D, Vézin P, Laporte S, et al. Will automated driving technologies make today's effective restraint systems obsolete? *Am J Public Health* 2017; **107**:1590–2.
124. Taylor JR, Twomey LT. Acute injuries to cervical joints: an autopsy study of neck sprain. *Spine* 1993; **18**: 1115–22.
125. Thorbole CK. Dangers of seatback recline in a moving vehicle: how seatback recline increases the injury severity and shifts injury pattern. In: *ASME 2015 International Mechanical Engineering Congress and Exposition, Volume 3: Biomedical and Biotechnology Engineering*. Houston, TX, USA, 2015.
126. Untaroiu CD, Adam TJ. Performance-based classification of occupant posture to reduce the risk of injury in a collision. *IEEE T Intell Transp* 2013; **14**:565–73.
127. Prasad VS, Schwartz A, Bhutani R, et al. Characteristics of injuries to the cervical spine and spinal cord in polytrauma patient population: experience from a regional trauma unit. *Spinal Cord* 1999; **37**:560–8.
128. Van Ee C, Nightingale R, Camacho D, et al. Tensile properties of the human muscular and ligamentous cervical spine. *Stapp Car C* 2000; **44**:85–102.
129. Vasavada AN, Brault JR, Siegmund GP. Musculotendon and fascicle strains in anterior and posterior neck muscles during whiplash injury. *Spine* 2007; **32**:756–65.
130. Viano D. Crashworthiness and biomechanics. In: *Euromotor Course*. Sweden: Gothenburg, 2001, 11–3.
131. Viano DC. *Role of the Seat in Rear Crash Safety*. Warrendale, PA: Society of Automotive Engineers, 2002.
132. Viano DC, Davidsson J. Neck displacements of volunteers, BioRID P3 and Hybrid III in rear impacts: implications to whiplash assessment by a neck displacement criterion (NDC). *Traffic Inj Prev* 2002; **3**:105–16.
133. Wang K, Deng Z, Wang H, et al. Influence of variations in stiffness of cervical ligaments on C5–C6 segment. *J Mech Behav Biomed* 2017; **72**:129–37.
134. Watanabe Y, Ichikawa H, Kayama O, et al. Influence of seat characteristics on occupant motion in low-speed rear impacts. *Accident Anal Prev* 2000; **32**:243–50.
135. Wiklund K, Larsson H. Saab Active Head Restraint (SAHR) - seat design to reduce the risk of neck injuries in rear impacts. *SAE Transactions* 1998; **107**:620–8.
136. Yamada H, Evans FG. *Strength of Biological Materials*. Baltimore: Williams & Wilkins, 1970.
137. Yamada K, Gotoh M, Kitagawa Y, et al. Simulation of occupant posture change during autonomous emergency braking and occupant kinematics in frontal collision. In: *Proceedings of the 2016 IRCOBI Conference*. Spain: Malaga, 2016, 261–74.
138. Yang R, Guo L, Huang L, et al. Epidemiological characteristics of traumatic spinal cord injury in Guangdong, China. *Spine* 2017; **42**:E555–61.

139. Yoganandan N, Cusick JF, Pintar FA, et al. Whiplash injury determination with conventional spine imaging and cryomicrotomy. *Spine* 2001; **26**:2443–8.
140. Yoganandan N, Kumaresan S, Pintar FA. Geometric and mechanical properties of human cervical spine ligaments. *J Biomech Eng* 2000; **122**:623–9.
141. Yoganandan N, Pintar FA, Cusick JF. Biomechanical analyses of whiplash injuries using an experimental model. *Accident Anal Prev* 2002; **34**:663–71.
142. Yoganandan N, Ray G, Pintar FA, et al. Stiffness and strain energy criteria to evaluate the threshold of injury to an intervertebral joint. *J Biomech* 1989; **22**:135–42.
143. Yu CY, Tsai KH, Hu WP, et al. Geometric and morphological changes of the intervertebral disc under fatigue testing. *Clin Biomech* 2003; **18**:3–9.
144. Zakrasek EC, Creasey G, Crew JD. Pressure ulcers in people with spinal cord injury in developing nations. *Spinal Cord* 2015; **53**:7–13.
145. Zhang X, Zhou Q. An energy-absorbing sliding seat for reducing neck injury risks in rear impact—analysis for prototype built. *Traffic Inj Prev* 2016; **17**:313–9.
146. Zhou S, Guo L, Zhang S, et al. Study on cervical spine injuries in vehicle side impact. *Mech Eng* 2010; **4**:29–35.